Effects of Increased Caloric Intake on Resting Metabolic Rate and Respiratory Quotient during a Ketogenic Diet: A Six-Month Case Study

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ABSTRACT

Stephenson S, Bolyard C, Stover S. Effects of Increased Caloric Intake on Resting Metabolic Rate and Respiratory Quotient during a Ketogenic Diet: A Six-Month Case Study. JEPonline 2018;21(5):64-72. A ketogenic diet is characterized by high fat content and very low carbohydrate content. It is associated with favorable anthropometric and cardiovascular outcomes. A CrossFit-trained male was on a high protein ketogenic diet for six months. Midway through the study, he increased his daily energy intake by 1000 kcal. Anthropometric, cardiovascular, and metabolic impacts were determined before and after the caloric increase. Resting metabolic rate (RMR) was measured by indirect calorimetry, and respiratory quotient (RQ) was calculated as the ratio of CO₂ production to O₂ consumption. Prior to the caloric increase, the subject exhibited an elevation in RMR and a dramatic decline in RQ, indicating increased utilization of fat as metabolic fuel. Following the caloric increase, compensatory adjustments for elevated body weight led to fluctuations in RMR and a significant increase in RQ, which indicate a shift to carbohydrate utilization.

Key words: Ketogenic Diet, Respiratory Quotient, Resting Metabolic Rate
INTRODUCTION

Lipids provide substantial energy during submaximal exercise (26). However, as exercise intensity increases, carbohydrate (CHO) oxidation increases as lipid oxidation decreases. The point at which lipid oxidation reaches its peak is referred to as maximum fat oxidation (MFO). Exercise intensities that exceed MFO oxidize CHO in greater proportion (5). It has been demonstrated that MFO can be increased with endurance training (1). The mitochondrial enzymes citrate synthase and 3-hydroxyacyl-CoA dehydrogenase (HAD) are upregulated in response to training (19). Citrate synthase increases the rate of the Krebs cycle, while HAD elevates the rate of beta-oxidation (25). Diets that incorporate higher proportions of a specific macronutrient promote the oxidation of that macronutrient (27). High fat diets increase beta-oxidation at rest and during moderate exercise (16). High fat diets also increase levels of intramuscular triglyceride, while decreasing muscle glycogen levels (28). However, high intensity exercise will still eclipse MFO and rely on glycolysis for ATP synthesis. Thus, substrate utilization is dictated by exercise intensity, regardless of training status or diet (14).

Ketone bodies (KBs) are an alternative to the usual fat and CHO-based fuel sources. KBs, which are produced in the liver during prolonged fasting or starvation, attenuate peripheral glucose utilization and promote lipolysis in adipose tissue (22). Ketogenesis, the production of KBs, is an adaptive response that facilitates survival during an energy crisis by providing a substrate for brain cells that cannot utilize free fatty acids as a fuel source (10). Ketogenesis liberates acetoacetate (AcAc), which is reduced to beta-hydroxybutyrate (bHB) in an NADH-requiring reaction catalyzed by 3-hydroxybutyrate dehydrogenase (BDH). Once bHB enters the mitochondrial matrix, it is oxidized to AcAc by BDH. Subsequent reactions generate two molecules of acetyl CoA, which are incorporated into the Krebs cycle (10).

A ketogenic diet (KD) is characterized by high fat, moderate protein, and very low CHO content. Such a diet promotes a state of ketosis, an increase in fat utilization resulting in an accumulation of KBs, and can produce favorable cardiometabolic outcomes, including stable decreases in body weight, body mass index (BMI), body fat, blood glucose, glycated hemoglobin (A1C), LDL cholesterol, and blood pressure (7). Furthermore, a KD does not seem to compromise aerobic endurance or explosive strength performance (20). A study with well-trained athletes demonstrated that prolonged ketosis produces an adaptation effect, allowing free fatty acids to become the primary metabolic fuel during moderate exercise (28). However, high intensity exercise suppresses lipolysis and increases glycolysis, promoting the utilization of CHO (8).

During periods of energy deficit, perhaps due to weight loss, energy expenditure may be reduced by slowing the resting metabolic rate (RMR). This metabolic adaptation is referred to as adaptive thermogenesis (11). RMR is the primary contributor to total energy expenditure, responsible for up to 75% of daily expenditure. Consequently, a chronic reduction in RMR could promote weight gain over time (2). A recent study in obese patients indicated that a KD did not decrease RMR even though it produced significant weight loss. The authors speculated that the preservation of lean body mass might have been responsible for the lack of adaptive thermogenesis (12).
The current study examined the case of a CrossFit-trained male on a high protein ketogenic diet (HPKD). Midway through the six-month study, he increased his daily energy intake by 1000 kcal. We recorded anthropometric (body weight, body fat, and BMI), cardiovascular (heart rate and blood pressure) and metabolic (RMR and respiratory quotient) adaptations to the increased caloric intake.

METHODS

Subject
The Institutional Review Board of Davis & Elkins College approved this study. The subject was a 57 yr old male, taking no medications, who began a HPKD in January of 2018. At the onset of the study, the subject had been on the diet for approximately 2 wks. He was not interested in losing weight, but wanted to add and maintain muscle mass. The HPKD allowed him to obtain roughly 95% of his daily energy intake from proteins and fats. Only about 5% came from CHO. For the first three months of the study, he consumed an average of 2800 kcal-day\(^{-1}\). In the middle of April, he was determined to put on more lean body mass, so he increased his daily intake to 3800 kcal-day\(^{-1}\). He maintained the increased caloric intake for the next three months. The subject had been training an average of 5 d-week\(^{-1}\) at a CrossFit gym for ~2 yrs and continued his training throughout the study. CrossFit training involved constantly varied functional movements performed at high intensity (9). To assess daily physical activity, the subject wore a wristband-embedded, fitness-tracking device (Fitbit Charge 2, Fitbit Inc., San Francisco, CA).

Procedures
The subject signed a consent form and agreed to fast and drink only water for at least 8 hrs prior to each data collection visit. Anthropometric, cardiovascular (CV), and metabolic data were collected at ~1 month intervals from mid-February to mid-July. All collections took place between 7:30 a.m. and 8:30 a.m. Body weight and body fat data were obtained using a digital scale capable of measuring electrical impedance (Fitbit Aria, Fitbit Inc., San Francisco, CA). BMI was determined by entering height and weight values into an online calculator provided by the National Heart, Lung, and Blood Institute (18). Resting heart rate (HR) and blood pressure were determined via fingertip pulse oximeter and sphygmomanometer, respectively. RMR was measured by indirect calorimetry. A metabolic cart and canopy system (COSMED Quark Cardiopulmonary Exercise Testing System, COSMED, Rome, Italy) were used to analyze gas exchange, allowing measurement of actual RMR and calculation of predicted RMR via the Harris-Benedict equation: 
\[
RMR = 66.473 + 5.003 \times \text{height} + 13.75 \times \text{weight} - 6.75 \times \text{age}.
\]
Respiratory quotient (RQ) is the ratio of CO\(_2\) exhaled to the amount of O\(_2\) consumed. Measured gas exchange data were used to calculate RQ and estimate the utilization of fat and CHO as fuel sources. The subject rested quietly in a supine position under the canopy for 20 min. The first 5 min served as an acclimation to steady state, a period when the average minute VO\(_2\) and VCO\(_2\) changes by less than 10% and the average RQ changes by less than 5%. Only data from the final 15 min of the test were used to determine RMR and RQ.

RESULTS

Fitness tracker data indicated that the subject averaged approximately 9800 steps-day\(^{-1}\) for the six months of the study. Anthropometric and CV data are presented in Table1.
weight and BMI peaked in month 4, ~30 days after the increase in caloric intake. There is no body fat data for the first month, as the subject had yet to synchronize his fitness tracker with the digital scale. With the exception of systolic (SBP) and diastolic (DBP) blood pressure, which remained relatively stable, all values were higher in the second half of the study.

Table 1. Anthropometric and Cardiovascular Data.

<table>
<thead>
<tr>
<th>Data Collection Points</th>
<th>Body Weight (kg)</th>
<th>Body Fat (%)</th>
<th>BMI</th>
<th>HR (beats·min⁻¹)</th>
<th>SBP (mmHg)</th>
<th>DBP (mmHg)</th>
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<tbody>
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<td>1</td>
<td>82.1</td>
<td>NA</td>
<td>22.6</td>
<td>48</td>
<td>117</td>
<td>80</td>
</tr>
<tr>
<td>2</td>
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<td>18.1</td>
<td>22.7</td>
<td>50</td>
<td>115</td>
<td>75</td>
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<tr>
<td>3</td>
<td>83.5</td>
<td>18.6</td>
<td>23.0</td>
<td>54</td>
<td>115</td>
<td>78</td>
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<tr>
<td>4</td>
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<td>19.0</td>
<td>23.7</td>
<td>61</td>
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<td>19.1</td>
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<td>18.9</td>
<td>23.4</td>
<td>58</td>
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</tr>
</tbody>
</table>

BMI, Body Mass Index; HR, Heart Rate; SBP, Systolic Blood Pressure; DBP, Diastolic Blood Pressure.

Measured RMR was highest in months 3 and 4, declined to predicted levels in month 5, and rebounded in the final month of the study (Figure 1). RQ decreased steadily in the first half of the study, but rebounded in the second half (Figure 2).

Figure 1. Predicted and Measured Resting Metabolic Rate (RMR).
DISCUSSION

Months 1-3
In a previous study utilizing fitness trackers, we characterized 8000 to 12000 steps·day⁻¹ as moderate exercise (4). While our subject’s fitness tracker data put him in the moderate exercise range, it did not take into account the intensity of his CrossFit training. A recent study of CrossFit trainees by Kephart et al. (15) reported no significant changes to RMR or RQ after 3 months on a KD. However, our case subject exhibited an elevated RMR in the 3rd month, as well as a steady decline in RQ over the 1st 3 months of his HPKD (Figures 1 and 2). The subject was consuming ~2800 kcal·day⁻¹ during the first half of the study, and a significant portion of those calories came from protein. The high protein content of the HPKD, in conjunction with substantial physical training, might have promoted weight gain by increasing lean body mass. The subject gained 1.4 kg (3.1 lbs) over the 1st 3 months (Table 1). High levels of physical activity coupled with high-energy intake are associated with increased RMR (21) and could potentially explain the bump in RMR at month 3.

The ratio of CO₂ exhaled to the amount of O₂ consumed, RQ, indicates which macronutrients are being utilized as different energy pathways are used for different macronutrients. A value of 0.7, for example, would indicate that fats are the primary fuel source, while a value of 1.0 would indicate that CHO is the favored fuel. Over the 1st 3 months, the subject relied more and more on fat utilization, and his RQ decreased from 0.92 to 0.77 (Figure 2). In response to the very low CHO intake, fat metabolism increased from an estimated 28% in the first month to 79% in month 3. Previous research demonstrated a similar decrease in RQ for overweight and obese men on a KD (13).

Months 4-6
In the second half of the study, our subject increased his caloric intake to approximately 3800 kcal·day⁻¹. The subject gained another 2.5 kg (5.5 lbs) of body weight in the 1st month after the caloric increase, but lost some of the weight gain, 0.9 kg (2.0 lbs), by the end of the study (Table 1). The increased energy intake along with intense physical training could be

![Figure 2. Changes in Respiratory Quotient (RQ).](image-url)
responsible for the small increase in RMR in month 4. Furthermore, it has been suggested that a slight increase in RMR can be caused by elevated hepatic $O_2$ consumption proportional to the rate of ketogenesis (23). A previous study demonstrated that an increase in fat intake could produce an elevation in resting HR, as metabolism increases to adjust to fat utilization (3). Our subject’s increased caloric intake included a substantial increase in fat intake, which could explain his slightly elevated resting HR in the 2nd half of the study (Table 1). The subsequent drop in RMR might have been due to a decrease in gluconeogenesis as the brain shifted away from glucose utilization and toward more KB utilization (6). The increased fat intake during the last 3 months of the study would have resulted in more ketogenesis. It has also been suggested that a decrease in insulin production could suppress RMR (17). We think that was unlikely to be a factor for our subject, given that his CHO intake increased proportionately in the 2nd half of the study. However, we cannot confirm this, as we did not analyze insulin levels. RMR rebounded in the last month of the study. Apparently, the subject had finally adjusted to the increased caloric intake, and his RMR returned to a level comparable to that of month 3, which was well above predicted levels (Figure 1).

The subject’s RQ was elevated dramatically following the caloric increase, eventually reaching a value comparable to that of month 1 (Figure 2). A high RQ is associated with an increase in body weight and fat mass (24). Our subject experienced a net gain of 1.6 kg (3.5 lbs) in the second half of the study, and a net gain of 3.0 kg (6.6 lbs) for the full 6 months (Table 1). By the end of the study, fat utilization was estimated to be at 32%.

CONCLUSION

Initially, the HPKD promoted an increase in RMR and a decrease in RQ, as the subject adjusted to very low CHO intake. Following the increase in overall caloric intake, metabolic compensation for an increased body weight resulted in fluctuations in RMR and an increase in RQ.

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REFERENCES


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